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Gingival and periodontal diseases in children

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ABSTRACT

Periodontal diseases, one of the most important health problems in societies, affect children as well as adults. Gingivitis is more common in childhood than periodontitis. Bone and attachment loss in children is not common, but may be associated with a systemic disease or an isolated dental condition. Early diagnosis and treatment of periodontal diseases in childhood and identification of patients at risk are very important in terms of preventing diseases that may occur in the future.

Keywords: Child, gingivitis, periodontal diseases, periodontitis

INTRODUCTION

Periodontal diseases in adults usually begin in early childhood. The role of dentists in the early diagnosis and treatment of periodontal diseases is very important.[1] If left untreated, these diseases seen in children negatively affect the child's nutrition and development.[2]

There are significant differences between periodontal tissues in childhood and adults.[3]

Periodontium in Primary Dentition

In the milk dentition period, the gums show some differences compared to the adult gums. The tissues are pale pink. The attached gingiva in children is less keratinized than the attached gingiva in adults. The thinness of the keratinized layer causes the underlying veins to be more visible. Stippling pattern occurs around 3 years of age and is detected in 56% of children aged 3-10 years. Little difference has been reported between the maxillary and mandibular arches or between boys or girls during childhood.[4]

Figure 1. Normal gingival appearance in a 5-year-old child.[1]

Differences Between Periodontal Structures in Children and Adults

In children, the interdental gingiva is wide in the buccolingual direction and narrow in the mesiodistal direction. In the interproximal areas, the papilla is observed as flattened. It is consistent with the morphology of the primary dentition. The structure and composition of the gums are similar to those of adults. The consistency of the gingiva is softer, the gingival margin is thick and roll-shaped. Although the gingiva is pale pink, it appears more red during the teething period. It is seen that the depth of the probing pocket is shallower in primary teeth compared to permanent teeth. Depth increases posteriorly and is approximately 1-2 mm.[3,4]

The width of the attached gingiva on the vestibule surfaces of anterior teeth is greater than that of posterior teeth. While the width of the buccal surfaces decreases from anterior to posterior, some data indicate a narrowing in the region of the canine teeth. On lingual surfaces, the situation is reversed; There is an increase in width from anterior to posterior. During the transition to permanent dentition, gingival width increases with age. The junctional epithelium is thicker in primary teeth. Thicker epithelium reduces the passage of bacteria and their toxins.[4]

Characteristics of Periodontal Tissues in Childhood

Radiographically, the lamina dura is more prominent, and the periodontal ligament space is wider. In children, the thickness of the periodontal membrane is increased. The density and amount of these fibers, which are irregular and loose, are less. Cementum is thinner and less calcified in primary teeth. In children, the alveolar bone is thinner. The bone marrow spaces are wider. The amount of calcification is less and the lymph and blood vessels are more. The alveolar crest is flatter, 1-2 mm from the enamel-cementum boundary. In children, alveolar bone contains more organic matter.[5,6]

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EPIDEMIOLOGY

In developed countries, the incidence of gingivitis in children aged 6-11 is around 73%. This rate increases from 6 to 11 years of age.[7] In many studies, it has been shown that there is a significant increase in the prevalence of gingivitis with adolescence.[8] It is observed that there is an increase of 50-99% in the incidence of gingivitis during adolescence.[8] It has been reported that the prevalence of gingivitis is lower among girls than boys and this is probably related to oral hygiene.[9]

CLASSIFICATION OF PERIODONTAL DISEASES IN CHILDREN AND ADOLESCENTS

The clinical classification of periodontal diseases in children and adolescents determined by the American Academy of Periodontology is as follows;[10]

1. Gingival Diseases
   - Gingival diseases induced by dental plaque
   - Gingival diseases not induced by dental plaque
2. Aggressive periodontitis
3. Chronic periodontitis
4. Periodontitis as a manifestation of systemic diseases
5. Necrotizing periodontal diseases

GINGIVAL DISEASES

Gingival Diseases Due to Dental Plaque

The most common periodontal infection among children and adolescents is plaque-induced chronic gingivitis. Studies have reported a 70% incidence in children after the age of seven. Swelling, bleeding and redness in the marginal gingiva are the first signs. Although the progression of these diseases to periodontitis is rare, the treatment of gingival diseases in children is important. Dental plaque resulting from poor oral hygiene is the primary cause of gingivitis. There is no strong correlation between dental plaque scores and gingival index in children. The same oral hygiene conditions cause milder disease development in children compared with adults.[11,12] The presence of gingivitis in school children may be caused by different dietary habits, the presence of mixed dentition, incorrect and uncontrolled oral hygiene practices, and malocclusion.[13]

The severity of gingivitis in children is mild compared to adults. This difference in the severity of gingivitis may be related to the nature of the dental plaque, differences in the immune system and the difference in the periodontium with adults. Dental plaque in children contains less amount of periodontal pathogen. The decreased vascularity of the gingival connective tissue and the formation of a thicker barrier limit the severity of infections. The incidence of gingivitis increases with age. While the prevalence of gingivitis is at its lowest level in pre-school, it peaks in puberty, especially between the ages of 14-16. Hormonal factors have a serious effect on this increase.[14,15]

Clinical features: Inflammation is generally limited to the marginal gingiva and progresses in different directions over time. Changes occur in the color, size and consistency of the gums, and they resemble those in adults. As the disease progresses, which starts with redness, swelling and bleeding in the gums, chronic changes such as fibrotic tissue becoming and hyperplasia can be observed. In the absence of severe gingival hypertrophy or hyperplasia, there is no significant increase in pocket depth and bleeding. In histological evaluation; It has been shown that especially cellular inflammatory cells (polymorphonuclear leukocytes, mast cells, plasma cells) are infiltrated into the junctional epithelium and there is a loss of collagen in the regions.[4,16,17]

In children and young adults with similar levels of dental plaque, inflammatory reactions are more pronounced in adults. Predominance of T lymphocytes and fewer B lymphocytes and plasma cells in children makes the host response different. In children, due to the thicker junctional epithelial barrier, decreased permeability and becoming a more difficult barrier for bacterial toxins makes it difficult to develop inflammation.[18,19]

Eruption gingivitis: Gingival inflammation that occurs during tooth eruption is called eruption gingivitis. In fact, it occurs as a result of increased plaque accumulation in areas where milk teeth fall out and permanent teeth begin to erupt. The areas where the teeth start to erupt are very sensitive and the bristles of the brush cause irritation in these areas. It is quite difficult to maintain hygiene in these areas.[20]

In primary teeth, the increase in the depth of the gingival sulcus over time and the apical migration of the junctional epithelium facilitate the eruption of the tooth. During rubbing, degenerative changes occur in the junctional epithelium and the permeability of the epithelium increases.[20] Sulcus development is incomplete, the gingival margin is not keratinized. As a result of inflammatory changes, the normal form of the gingiva changes and a distinctive gingival enlargement image occurs.[4,8] During the eruption of the teeth, food entrapments may exacerbate the condition and pericoronal abscess or pericoronitis may develop in the erupting tooth. Providing effective plaque control will reduce these reactions occurring in the gingiva.[12]

Puberty gingivitis: Different factors, such as plaque scores, dental caries, tooth eruption, crowding, and mouth breathing, affect the severity and incidence of gingivitis in adolescents.[11] However, the severity and prevalence of gingivitis reaches its highest level as a result of the serious increase in steroid hormones between the ages of 9-14, which coincides with the pre-pubertal and pubertal period. This increase has a temporary effect on the condition of the gums and ends after adolescence.[4] Gums contain receptors with a high affinity for estrogen and progesterone. Estrogen receptors are located in the basal layer of the epithelium, spinous layer, connective tissue endothelium, and fibroblast cells in small vessels.[8] These hormones increase vascular permeability and may cause damage to the endothelium. In addition, it affects the migration of leukocytes to the inflamed tissue, the incorporation of the subgingival flora, and the formation of granulation tissue.[20]

The relationship between increased levels of steroid hormones in the circulation and gingivitis during adolescence was evaluated and it was observed that inflammation increased at an earlier age (10-13) in girls.[8] Estrogen and progesterone levels were found to be associated with an increase in *P. intermedius* rates during this period, and in vivo studies have shown that *P. intermedius* uses these hormones as a food source.[20]

Pubertal gingivitis is characterized by marked inflammation, bluish-red discoloration, edema, gingival

enlargement, and spontaneous bleeding, especially in the interdental areas.[8,16] In addition to an effective oral hygiene regimen at home, elimination of local factors and professional prophylaxis will be very effective.[16]

Gingival growths due to drug use: As a result of the use of cyclosporine, calcium channel blockers and phenytoin, gingival enlargement may occur and its prevalence in children is quite high. The clinical and microscopic features of growth induced by different drugs are similar. Growth begins in the interdental space and spreads to completely cover the marginal gingiva. In cases where the severity of the disease increases, the gingiva may enlarge to cover the incisal and occlusal surfaces. Gingival enlargement is common throughout the mouth, but more severe in the maxillary and mandibular anterior region. Gingival enlargement is usually seen in the area of the teeth, not in the edentulous areas. When the teeth are extracted, the growth disappears. Mucosal hyperplasia has also been reported in edentulous mouths, but is very rare. There is a direct relationship between the severity of growth and the amount of plaque. Removal of dental plaque and maintaining oral hygiene can reduce the severity of lesions.[21-23]

Growths cause aesthetic problems, chewing, speaking, driving problems, tissue traumas, secondary infections, and periodontal diseases. First of all, the attachments on the teeth should be removed and the patient should be taught to brush and floss correctly. Operations such as gingivectomy and gingivoplasty may be necessary to correct the gingival contours and ensure the full applicability of hygiene procedures. The patient and their relatives should be informed that the growths may recur after the surgical procedure, and the importance of an effective plaque control in controlling the process should be emphasized. Gingival enlargements tend to disappear spontaneously within a few months after discontinuation of the drug. A decision can be made to change the drug, if appropriate, by asking the patient for a medical consultation.[16,24]

Oral Lesions Independent of Plaque

Recurrent aphthous stomatitis, primary herpetic gingivostomatitis, recurrent herpes simplex infections, common oral candidiasis, angular cheilitis and geographic tongue are the most common oral lesions in children. The incidence of these lesions is approximately the same between children and adults.[24,25]

Primary herpetic gingivostomatitis: Primary herpetic gingivostomatitis is a viral disease that usually occurs acutely in early childhood and its causative agent is Herpes simplex virus (HSV) type-1. PHG often affects children younger than 10 years of age, more frequently in children aged 2 to 4 years, and then younger people aged 15-25.[16] In the vast majority of cases, 99%, there are no symptoms or the resulting symptoms are attributed to teething. In the remaining 1%, severe inflammation and ulceration of the lips and mucosa may develop. The disease has an incubation period of about 1 week. The clinical features of the disease are fever, headache, cervical lymphadenopathy, diffuse inflammation characterized by significant swelling, bleeding, and erythema in the marginal and attached gingiva. The vesicles burst, forming large ulcers. Patients have difficulty in chewing and swallowing due to severe pain. For this reason, it is very important to take plenty of fluids in order not to develop dehydration. PHG is an infectious disease that usually regresses spontaneously within 12-20 days.[24,25] Healing occurs spontaneously within 10-14 days without leaving a scar.

In the treatment of PHG, prevention of bacterial superinfections is important in terms of early healing of ulcerations. Therefore, dental plaque must be carefully removed.[26] For patients with herpetic gingivostomatitis, if acyclovir oral suspension therapy is administered at 15 mg/kg starting within the first 3 days, a significant reduction in the duration of contagiousness and symptoms in affected children has been reported.[16]

Candidiasis: It is caused by an overgrowth of *Candida albicans*, an opportunistic fungus found in the oral cavity and different parts of the body. The rate of C. albicans can reach 50%-80% in the total mouth fungus population. It has proteinase-containing strains that can invade the keratinized epithelium and is the most common fungal infection in the oral mucosa.[26] It is generally transmitted from the mother during birth to infants, and its incidence increases after long-term antibiotic or steroid use in older children. In addition, it is frequently seen in diabetes, hypoparathyroidism and chemotherapy patients. HIV-infected children and adolescents are prone to develop oral candidiasis.[16] Oral candidiasis is rarely seen on the gums in healthy individuals. It can be seen in different types in the oral mucosa, such as pseudomembranous candidiasis (thrush), erythematous candidiasis, plaque type, nodular.[26] Clinically, it is in the form of diffuse plaques in the form of whitish patches that can be easily removed from the mucosa with the help of a tool or cloth.[26] In topical treatment, antifungals such as nystatin, amphotericin B, and miconazole are applied.[24]

Recurrent herpes simplex: Primary infections are usually transmitted by direct contact with a lesion or with infected body fluids (saliva, exudates of active lesions). HSV Type 1 binds to specific surface receptors of cells and infects the oral mucosa. After primary infection, it settles in the ganglia of neurons via sensory and autonomic nerves. The virus specifically settles in the trigeminal ganglion, where it remains latent for life.[16,27] Reactivation of the virus in the sensory ganglion causes cutaneous and mucocutaneous recurrent herpetic infection. Reactivation may occur spontaneously or may be triggered by a number of factors.[16]

In general, HSV type-1 is responsible for infections in the orofacial region, and the virus is found at a rate of 30-60% in children. Primary HSV infections in children are usually asymptomatic or are too mild to be noticed. Recurrent infections occur as a result of triggering factors, such as sunlight, fatigue, emotional stress, trauma, fever, and immunosuppression. The most common secondary recurrent infections are herpes labialis.[28] Lesions typically occur at the mucocutaneous junction called the vermillion, especially on the lips. In this region, symptoms such as paresthesia, itching, pain, and burning sensation are observed in 46-60% of patients. Lesions rapidly become vesicular, then burst into ulcers or crusted pustules.[28,29]

Recurrent aphthous stomatitis (RAS): The most common form of RAS in childhood is minor aphthae.[28] Patients often refer to these ulcerative lesions as "cancers". RAS lesions can sometimes be 0.5 to 1.0 cm in diameter, or appear as oval and irregular ulcers ranging in size from 1 to 3 mm. Small lesions heal in 7 to 10 days, while larger lesions take weeks to heal with scarring.[16] Trauma, emotional stress, problems with

menstruation, nutrition, and endocrinopathies predispose to the emergence of RAS. Although there is no specific treatment for RAS, topical tetracycline, chlorhexidine gluconate or other mouthwashes, topical corticosteroids or immunomodulators can be used in the clinical management of the disease.[24,30]

Angular cheilitis: Inflammation begins at the corners of the lips. Over time, erosion, ulceration, fissure develops, and it is a very painful condition. *Candida albicans* and *Staphylococcus aureus* have been reported to cause angular cheilitis. Angular cheilitis and other oral candidiasis infections are very common in HIV-infected children. Factors such as immunodeficiency, vitamin B2 deficiency, decrease or loss of vertical dimension for various reasons, and trauma predispose to these infections.[16,24]

Geographic tongue (benign migratory glossitis): Geographic tongue is a benign inflammatory condition. It is characterized by a desquamation of superficial keratin and filiform papillae in the tongue. It affects approximately 1-2% of the population. Although its etiology is unknown, it has been suggested that it correlates with nutritional deficiencies and emotional stress. This condition is usually limited to the dorsum and lateral of the tongue. Sometimes the lesions become symptomatic and a burning sensation occurs in the tongue.[31,32] It is seen that the complaints are sometimes quite severe, and sometimes completely reduced. Patients with symptoms may be advised to stay away from acidic and spicy foods.[24] Topical or systemic antihistamines can be used in geographic tongue lesions.[16]

Periodontal diseases in childhood: Epidemiological studies have shown that all children and adolescents in the world are affected by different forms of gingivitis. Bone and attachment loss is not common in children. However, the incidence of gingivitis in children 7 years and older is more than 70%. In epidemiological studies conducted in the USA, severe attachment loss in one or more teeth in children and young adults was found to be approximately 0.2-0.5%.

When different forms of periodontal diseases are compared; chronic periodontitis is more likely to occur in adults, and aggressive periodontitis in children and adolescents.[12,33]

AGGRESSIVE PERIODONTITIS

Aggressive periodontitis is known for the clinical signs of periodontitis to appear at a very early age, to show a strong tendency to pass in the family, and to show a large amount of attachment and bone loss in a short time. Pathogenic microflora, abnormalities in host defense mechanism, phagocyte abnormalities and hypersensitive macrophage phenotype are the main causes of the disease. It is divided into subgroups as localized and generalized. The prevalence of the localized form, which is more common in young people compared to the generalized form, is between 0.1% and 15%. In the 14 -17 age group, the rate of generalized aggressive periodontitis is 0.13%.[16,34]

Localized Aggressive Periodontitis (LAgP)

The disease begins around puberty. In these patients, the loss of attachment in the interproximal areas should not exceed two teeth, except for the permanent first molars and incisors. Studies so far have shown that in most cases of LAgP coexisting *Aggregatibacter* (*Actinobacillus*) spp., *Bacteroides* spp. and *Eubacterium* spp.

It has also been proven that cases of LAgP are associated with defects in the function of neutrophils. In these patients, the host response to the infecting agent is very severe, increasing the extent of destruction.

Clinical examination of patients with Localized Aggressive Periodontitis (LAgP) shows that the amount of plaque and calculus is too low to be associated with the severity of inflammation. No clinical signs of systemic diseases are observed in patients.[24,35]

Generalized Aggressive Periodontitis (GAgP)

It mostly affects individuals under the age of 30. It usually affects the entire dentition and is known as a disease of young adults. Except for the permanent incisor and permanent first molars, there must be at least three other permanent teeth affected. There is no strong antibody response to the infecting agent as in LAgP. Affected teeth have a high proportion of *Porphyromonas gingivalis* in the subgingival flora. In addition, chemotaxis defects in neutrophils are observed in these patients.[1,36]

In the treatment of aggressive periodontitis, successful results are obtained with the use of systemic antibiotics in addition to detection, subgingival curettage, root surface straightening and periodontal surgeries.

Studies have shown that the use of tetracycline alone or in combination with metronidazole in aggressive periodontitis gives very successful results. Amoxicillin-metronidazole combination may be preferred in younger children because tetracycline causes discoloration in teeth.[37] It has been reported that LAgP patients respond quite well to the treatments, while GAgP patients do not give the desired response to both periodontal treatments and antibiotics. The characteristic structure of the pathogenic flora may necessitate the use of alternative antibiotics. When there is no response to standard treatments, laboratory tests should be performed by taking plaque samples from GAgP patients and pathogens resistant to routinely used drugs should be determined.

There are some retrospective studies suggesting that the presence of bone loss in primary teeth may be an early sign of localized aggressive periodontitis. Therefore, periodontal evaluation should be done carefully in children.[12,38,39]

CHRONIC PERIODONTITIS

Children are not as susceptible to this form of periodontitis as adults, but more rarely, it can affect children and adolescents. The disease usually progresses at a low or moderate rate. In some periods, there may be an exacerbation of destruction attacks. It is divided into two as localized or generalized according to the ratio of the affected areas. If the relevant areas are less than 30%, they are called localized, and if they are more, they are called generalized. clinical attachment loss of 1-2 mm; mild clinical attachment loss of 3-4 mm; moderate, losses of 5 mm and above; it is classified as severe.[12,40,41]

Before chronic periodontitis, the patient must have a history of gingivitis. However, it is very difficult to determine the exact stages in the transition from gingivitis to periodontitis. Factors such as an increase in pathological bacteria, immune system-related conditions, genetic susceptibility of patients may be possible causes.

The general approach to chronic periodontitis is to eliminate the existing inflammation, stop the progression

of the disease and provide the patient with a stable oral environment. Providing personal plaque control, periodontal initial treatments such as detection, subgingival curettage, root surface straightening, mouthwashes are important in the treatment of the disease.[34] Electric toothbrushes can be recommended for mentally or physically handicapped children and for children who cannot use brushes effectively.[4]

PERIODONTITIS AS A SIGN OF SYSTEMIC DISEASES

Hypophosphatasia, Papillon-Lefevre Syndrome, leukemias, Chediak-Higashi syndrome, histiocytosis X, Acrodynia, Down syndrome, AIDS, Leukocyte adhesion defect, neutropenia, insulin-dependent diabetes are systemic diseases that cause periodontitis in children.[16] Defects in neutrophils and immune cell functions play a role in increased susceptibility to periodontitis and other infections.[43] It can occur in localized or generalized forms. Severe bone loss is seen around the teeth in the affected areas in patients.[41]

The percentage of potent periodontopathogens such as *A. actinomycetemcomitans*, *P. intermedia*, *E. corrodens* and *Capnocytophaga* increased in the affected areas.[41]

Necrotizing Periodontal Diseases

The presence of acute pain in the interdental areas, as well as areas of necrosis and ulceration resembling a crater in the form of punch holes, are among the important symptoms. Easily bleeding pseudo-membranes cover the lesions. Spirochete invasion and presence of *P. intermedia* on necrotic surfaces have been shown in studies.[24] Systemic findings such as fever and lymphadenopathy may often accompany necrotizing periodontal diseases. Children may become prone to these diseases due to viral infections, nutritional deficiencies, various systemic diseases, fatigue and stress.[41] In the presence of systemic findings, it is recommended to use antibiotics such as metronidazole and penicillin.[42] However, it has been shown that the effect of mechanical debridement with ultrasonic devices is more positive and faster.[24]

CONCLUSION

Periodontal diseases are among the most common diseases affecting children and adolescents. It is well known that bacterial plaque plays a role in the primary etiology of periodontal diseases, but when evaluating periodontal diseases in patients affected by early-onset periodontitis, diseases that may affect the host's defense mechanism should definitely be considered.[42]

To best manage periodontal diseases, dentists should have sufficient knowledge of the diagnostic features, occurrence, prevalence, microbiology and treatment of these diseases.[34] Periodontal evaluation of routine dental check-ups in childhood will ensure early prevention of diseases that may occur in the future. If the disease is already present, early treatment, teaching the child to brush correctly, and increasing the family's awareness of oral hygiene will reduce the prevalence of the disease in societies.

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REFERENCES

1. Clerehugh V, Kindelan S. Guidelines for periodontal screening and management of children and adolescents under 18 years of age. *Br Soc Periodontol Br Soc Pediatr Dent.* 2012;1:1-25.
2. Akçakoca A, Meşeli S, Yıldırım H, Mungan N, Akyüz S, Kuru L. Çocuklarda beslenme ve ağız bakım alışkanlıkları ile etkileyen faktörlerin değerlendirilmesi. *Yeditepe Klinik.* 2021;17(1):54-61. 10.5505/yeditepe.2021.48403
3. Bimstein Enrique, Needleman Howard L, et al. Periodontal and gingival health and diseases. United Kingdom: Martin Dunitz; 2001. Chapter 2, The normal gingival and periodontium; p. 17–30.
4. Carranza FA. Gingival diseases in childhood. In: Newman MG, Takei HH, Klokkevold PR, editors. Carranza's Clinical Periodontology. 10th ed. Philadelphia: Saunders,2006; 404-410.
5. McDonald Ralph E, Avery David R, Dean Jeffrey A. Dentistry for Child and Adolescent. 8th ed. Missouri: Mosby; 2004. Chapter 20, Gingivitis and Periodontal Diseases: p.45-413.
6. Al-Ghutaimel H, Riba H, Al-Kahtani S, Al-Duhaimi S. Common periodontal diseases of children and adolescents. *Int J Dent.* 2014; 2014:850674. doi:10.1155/2014/850674
7. Ketabi M, Tazhibi M, Mohebrasool S. The prevalance and risk factors of gingivitis among the children referred to Isfahan Islamic Azad University (Khorasgan Branch) Dental School, In Iran. *Dent Res J.* 2006;3(1):33-36.
8. Pari A, Ilango P, Subbareddy V, Katamreddy V, Parthasarthy H. Gingival diseases in childhood - a review. *J Clin Diagn Res.* 2014;8(10):ZE01-ZE4. doi:10.7860/JCDR/2014/9004.4957
9. Eltas A Guler C, Uslu MÖ, Dündar S, Ersöz M, Eltas ŞD. Malatya şehir merkezi ve yakın beldelerde 7 - 14 yaş arasındaki çocuklarda periodontal durumun değerlendirilmesi. *Ata Diş Hek Fak Derg.* 2012; 2012(2): 108-113.
10. Ranney RR. Classification of periodontal diseases. *Periodontol 2000.* 1993;2:13-25. doi:10.1111/j.1600-0757.1993.tb00216.x
11. Stamm JW. Epidemiology of gingivitis. *J Clin Periodontol.* 1986;13(5):360-366. doi:10.1111/j.1600-051x.1986.tb01473.x
12. Califano JV; Research, Science and Therapy Committee American Academy of Periodontology. Position paper: periodontal diseases of children and adolescents. *J Periodontol.* 2003;74(11):1696-1704. doi:10.1902/jop.2003.74.11.1696
13. AlGhamdi AS, Almarghlani AA, Alyafi RA, Kayal RA, Al-Zahrani MS. Gingival health and oral hygiene practices among high school children in Saudi Arabia. *Ann Saudi Med.* 2020;40(2):126-135. doi:10.5144/0256-4947.2020.126
14. Matsson L. Development of gingivitis in pre-school children and young adults. A comparative experimental study. *J Clin Periodontol.* 1978;5(1):24-34. doi:10.1111/j.1600-051x.1978.tb01903.x
15. Bimstein E, Matsson L. Growth and development considerations in the diagnosis of gingivitis and periodontitis in children. *Pediatr Dent.* 1999;21(3):186-191.
16. Oh TJ, Eber R, Wang HL. Periodontal diseases in the child and adolescent. *J Clin Periodontol.* 2002;29(5):400-410. doi:10.1034/j.1600-051x.2002.290504.x
17. Nowzari H, Botero JE. Latin America: native populations affected by early onset periodontal disease. *J Calif Dent Assoc.* 2011;39(6):383-391.
18. Seymour GJ, Crouch MS, Powell RN. The phenotypic characterization of lymphoid cell subpopulations in gingivitis in children. *J Periodontal Res.* 1981;16(5):582-592. doi:10.1111/j.1600-0765.1981.tb02020.x
19. Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. *Lab Invest.* 1976;34(3):235-249.
20. Bimstein E, Matsson L. Growth and development considerations in the diagnosis of gingivitis and periodontitis in children. *Pediatr Dent.* 1999;21(3):186-191.
21. Seymour RA, Ellis JS, Thomason JM. Risk factors for drug-induced gingival overgrowth. *J Clin Periodontol.* 2000;27(4):217-223. doi:10.1034/j.1600-051x.2000.027004217.x
22. Chapple IL. Periodontal diseases in children and adolescents: classification, aetiology and management. *Dent Update.* 1996;23(5):210-216.

23. Mariotti A. Dental plaque-induced gingival diseases. *Ann Periodontol.* 1999;4(1):7-19. doi:10.1902/annals.1999.4.1.7
24. Çalışır M, Akpınar A. Çocuklarda ve adolesanlarda periodontal hastalıklar. *Cumhuriyet Üniv Diş Hek Fak Derg.* 2013;16:226-234. doi:10.21306/jids.2017.1.27
25. Main DM. Acute herpetic stomatitis: referrals to Leeds Dental Hospital 1978-1987. *Br Dent J.* 1989;166(1):14-16. doi:10.1038/sj.bdj.4806682
26. Lang NP, Lindhe J. Clinical periodontology and implant dentistry, 2 volume Set, 6th edition. *Clin Periodontol Implant Dent.* 2015;340-376.
27. Arduino PG, Porter SR. Herpes simplex virus type 1 infection: overview on relevant clinico-pathological features. *J Oral Pathol Med.* 2008;37(2):107-121. doi:10.1111/j.1600-0714.2007.00586.x
28. Odom RB, James WD, Berger TG. Viral diseases. In: Andrew's Diseases of the Skin: Clin Dermatol. Philadelphia: WB Saunders Company, 2000:473-525
29. Field EA, Brookes V, Tyldesley WR. Recurrent aphthous ulceration in children--a review. *Int J Paediatr Dent.* 1992;2(1):1-10. doi:10.1111/j.1365-263x.1992.tb00001.x
30. Straus SE, Schmader KE, Oxman MN. Varicella and herpes zoster. In: Fitzpatrick TB; Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, eds. Fitzpatrick's Dermatology in General Medicine. New York, NY: McGraw-Hill Book Company;2003:2070-2085.
31. Straus SE, Schmader KE, Oxman MN. Varicella and herpes zoster. In: Fitzpatrick TB; Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, eds. Fitzpatrick's Dermatology in General Medicine. New York, NY: McGraw-Hill Book Company;2003:2070-2085.
32. Kleinman DD, Swango PA, Pindborg JJ. Epidemiology of oral mucosal lesions in United States school children: 1986–1987. *Community Dent Oral Epidemiol. 1994*;22:243–253.
33. Califano JV; Research, Science and Therapy Committee American Academy of Periodontology. Position paper: periodontal diseases of children and adolescents. *J Periodontol.* 2003;74(11):1696-1704. doi:10.1902/jop.2003.74.11.1696
34. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol.* 1999;4(1):1-6. doi:10.1902/annals.1999.4.1.1
35. Durmuşlar S, Akcabaş B. Çocuklarda ve adolesanlarda periodontal hastalıklar ve erişkinliğe etkisi. *J Int Dent Sci.* 2017;3:8-16.
36. Wilson ME, Zambon JJ, Suzuki JB, Genco RJ. Generalized juvenile periodontitis, defective neutrophil chemotaxis and *Bacteroides* gingivalis in a 13-year-old female. *J Periodontol.* 1985;56:457-463.
37. Al-Ghutaimel H, Riba H, Al-Kahtani S, Al-Duhaimi S. Common periodontal diseases of children and adolescents. *Int J Dent.* 2014;2014:850674
38. Van Winkelhoff AJ, Rams TE, Slots J. Systemic antibiotic therapy in periodontics. *Periodontol.* 2000 1996;10:45-78.
39. Van Winkelhoff AJ, Graaff J. Microbiology in the management of destructive periodontal disease. *J Clin Periodontol.* 1991; 18:406-410.
40. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: I. Response to 4 therapeutic modalities. *J Periodontol.* 1996;67:93-102
41. Chauhan, Vivek Singh, et al. "Gingival and periodontal diseases in children and adolescents." *J Dent Allied Sci.* 2012;1:26-29.
42. Johnson BD, Engel D. Acute necrotizing ulcerative gingivitis. A review of diagnosis, etiology and treatment. *J Periodontol.* 1986;57(3):141-50.
43. Hemalatha R, Leelarani V, Shakila KR. Gingival and periodontal diseases in childhood-a review. *J Evolution Med Dent Sci.* 2017;6:983-987.